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Luteoviruses

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Glossary

Hemocoel The primary body cavity of most arthropods that contains most of the major organs and through which the hemolymph circulates.
Hemolymph A circulatory fluid in the body cavities (hemocoels) and tissues of arthropods that is analogous to blood and/or lymph of vertebrates.

Introduction

Viruses of the family *Luteoviridae* (luteovirids) cause economically important diseases in many monocotyledonous and dicotyledonous crop plants, including barley, wheat, potatoes, lettuce, legumes, and sugar beets. Yield reductions as high as 30% have been reported in epidemic years, although in some cases crops can be totally destroyed. Diseases caused by the viruses were recorded decades and even centuries before they were associated with the causal viruses. In many cases, the stunted, deformed, and discolored plants that result from luteovirid infection were thought to be the result of abiotic factors, such as mineral imbalances or stressful environmental conditions, or of other biotic agents. This, along with their inabilities to be transmitted mechanically, delayed the initial association of the symptoms with plant viruses. For example, curling of potato leaves was first described in Lancashire, UK, in the 1760s, but was not recognized as a specific disease of potato until 1905 and to be caused by an aphid-transmitted virus until the 1920s. The causal agent, potato leaf roll virus (PLRV), was not purified until the 1960s. Similarly, widespread disease outbreaks in cereals, probably caused by barley yellow dwarf virus (BYDV), were noted in the United States in 1907 and 1949. In 1951, a virus was proposed as the cause. Other diseases caused by luteovirids, like sugarcane yellow leaf, which is caused by sugarcane yellow leaf virus (ScYLV), were not described until the 1990s.

Taxonomy and Classification

Members of the family *Luteoviridae* were first grouped because of their common biological properties. These properties included persistent transmission by aphid vectors and the induction of yellowing symptoms in many infected host plants. 'Luteo' comes from the Latin *luteus*, which translates as yellowish. All luteovirids have small (*c.* 25 nm diameter) icosahedral particles, composed of one major and one minor protein component and a single molecule of positive-sense single-stranded RNA of approximately 5600 nt in length.

The family *Luteoviridae* is divided into three genera – *Luteovirus*, *Polerovirus* (derived from potato leaf roll), and *Enamovirus* (derived from pea enation mosaic) – based on the arrangements, sizes, and phylogenetic relationships of the predicted amino acid sequences of the open reading frames (ORFs). In some plant virus families, a single gene can be used to infer taxonomic and phylogenetic relationships. Within the family *Luteoviridae,* however, different taxonomic relationships can be predicted depending on whether sequences of the replicase (ORF2) or coat protein (CP; ORF3) genes are analyzed (**Figure 1**). ORFs 1 and 2 of the luteoviruses are most closely related to the polymerase genes of viruses of the family *Tombusviridae*, while ORFs 1 and 2 of the poleroviruses and enamoviruses are related to those of the genus *Sobemovirus.* These polymerase types are distantly related in evolutionary terms. Consequently, it has been suggested that luteovirid genomic RNAs arose by recombination between ancestral genomes containing the CP genes characteristic of the family *Luteoviridae* and genomes containing either of the two

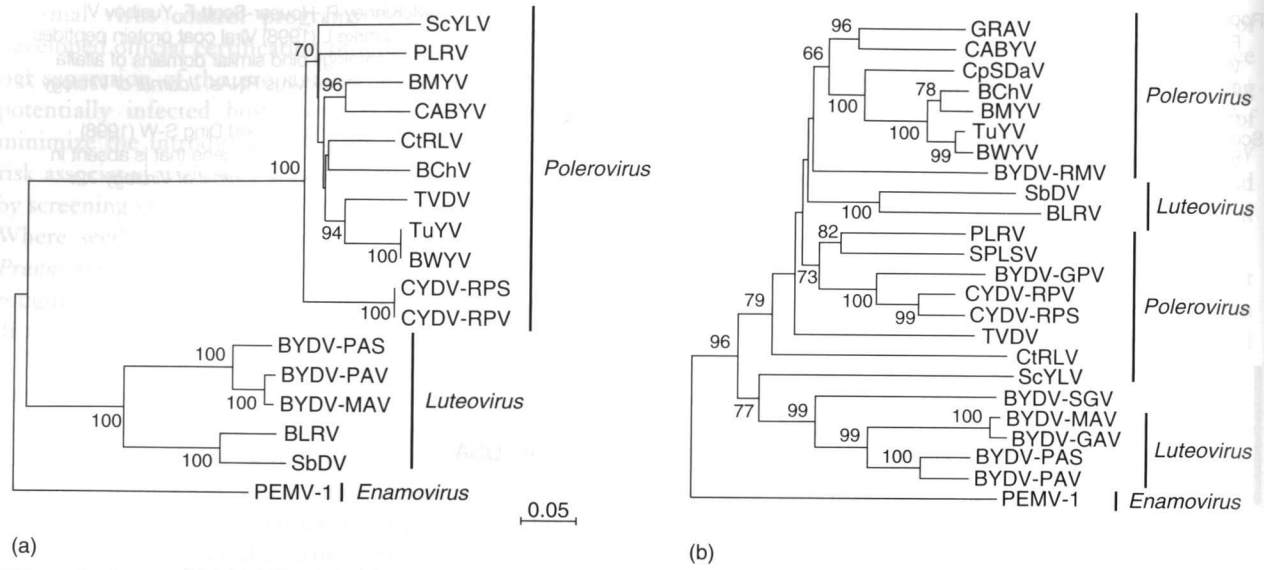

Figure 1 Phylogenetic relationships of the predicted amino acid sequences of the (a) RNA-dependent RNA polymerase (ORF2) and (b) major capsid protein (ORF3). When predicted amino acid sequences from ORF2 are used to group virus species the genera form three distinct groups. Using predicted amino acid sequences from ORF3, species of the genera *Luteovirus* and *Polerovirus* are intermingled in the tree. The resulting consensus trees from 1000 bootstrap replications are shown. The numbers above each node indicate the percentage of bootstrap replicates in which that node was recovered. For virus abbreviations, see **Table 1**.

polymerase types. For taxonomic purposes, the polymerase type has been the primary determinant in assigning a virus to a genus. For this reason, viruses for which only CP sequences have been determined have not been assigned to a genus. The current members of the family are listed in **Table 1**. The genus *Luteovirus* contains five species, and the *Polerovirus* genus has nine species. The genus *Enamovirus* contains a single virus, pea enation mosaic virus 1 (PEMV-1). The family also contains 11 virus species that have not been assigned to a genus. Of these, recently determined sequences of genomic RNAs of BYDV-GAV and carrot red leaf virus (CtRLV) suggest that BYDV-GAV is a strain of BYDV-PAV and that CtRLV is a unique species in the genus *Polerovirus*.

Virion Properties

The sedimentation coefficients $S_{20,w}$ (in Svedberg units) for luteoviruses and poleroviruses range from 106S to 127S. Buoyant densities in CsCl are approximately 1.40 g cm^{-3}. The particles formed as result of the mixed infections by PEMV-1 and PEMV-2 sediment as two components. The $S_{20,w}$ are 107–122S for B components (PEMV-1) and 91–106S for T components (PEMV-2, an umbravirus). Virions are moderately stable and are insensitive to treatment with chloroform or nonionic detergents, but are disrupted by prolonged treatment with high concentrations of salts. Luteovirus and polerovirus particles are insensitive to freezing.

Virion Structure and Composition

All members of the *Luteoviridae* have nonenveloped icosahedral particles with diameters of 25–28 nm (**Figure 2**). Capsids are composed of major (21–23 kDa) and minor (54–76 kDa) CPs, which contain a C-terminal extension to the major CP called the readthrough domain (RTD). According to X-ray diffraction and molecular mass analysis, virions consist of 180 protein subunits, arranged in $T = 3$ icosahedra. Virus particles do not contain lipids or carbohydrates.

Virions contain a single molecule of single-stranded positive-sense RNA of 5300–5900 nt. The RNAs do not have a 3′ terminal poly(A) tract. A small protein (VPg) is covalently linked to the 5′ end of polerovirus and enamovirus genomic RNAs. Cereal yellow dwarf virus RPV (CYDV-RPV) also encapsidates a 322 nt satellite RNA that accumulates to high levels in the presence of the helper virus. Complete genome sequences have been determined for 17 members of the *Luteoviridae* (**Table 1**). For several viruses, genome sequences have been determined from multiple isolates.

Genome Organization and Expression

Genomic RNAs of luteovirids contain five to eight ORFs (**Figure 3**). ORFs 1, 2, 3, and 5 are shared among all members of the *Luteoviridae*. Luteoviruses lack ORF0. Enamoviruses lack ORF4. The luteo- and polerovirus

Table 1 Virus members in the family *Luteoviridae*

Genus	*Virus*	*Abbreviation*	*Accession number*
Luteovirus	Barley yellow dwarf virus – MAV	BYDV-MAV	NC_003680[a]
	Barley yellow dwarf virus – PAS	BYDV-PAS	NC_002160
	Barley yellow dwarf virus – PAV	BYDV-PAV	NC_004750
	Bean leafroll virus	BLRV	NC_003369
	Soybean dwarf virus	SbDV	NC_003056
Polerovirus	Beet chlorosis virus	BChV	NC_002766
	Beet mild yellowing virus	BMYV	NC_003491
	Beet western yellows virus	BWYV	NC_004756
	Cereal yellow dwarf virus – RPS	CYDV-RPS	NC_002198
	Cereal yellow dwarf virus – RPV	CYDV-RPV	NC_004751
	Cucurbit aphid-borne yellows virus	CABYV	NC_003688
	Potato leafroll virus	PLRV	NC_001747
	Turnip yellows virus	TuYV	NC_003743
	Sugarcane yellow leaf virus	ScYLV	NC_000874
Enamovirus	Pea enation mosaic virus 1	PEMV-1	NC_003629
Unassigned	Barley yellow dwarf virus – GAV	BYDV-GAV	NC_004666
	Barley yellow dwarf virus – GPV	BYDV-GPV	L10356
	Barley yellow dwarf virus – RMV	BYDV-RMV	Z14123
	Barley yellow dwarf virus – SGV	BYDV-SGV	U06865
	Carrot red leaf virus	CtRLV	NC_006265
	Chickpea stunt disease associated virus	CpSDaV	Y11530
	Groundnut rosette assistor virus	GRAV	Z68894
	Indonesian soybean dwarf virus	ISDV	
	Sweet potato leaf speckling virus	SPLSV	
	Tobacco necrotic dwarf virus	TNDV	
	Tobacco vein distorting virus	TVDV	AJ575129

[a]Accession numbers beginning with NC_ represent complete genomic sequences.

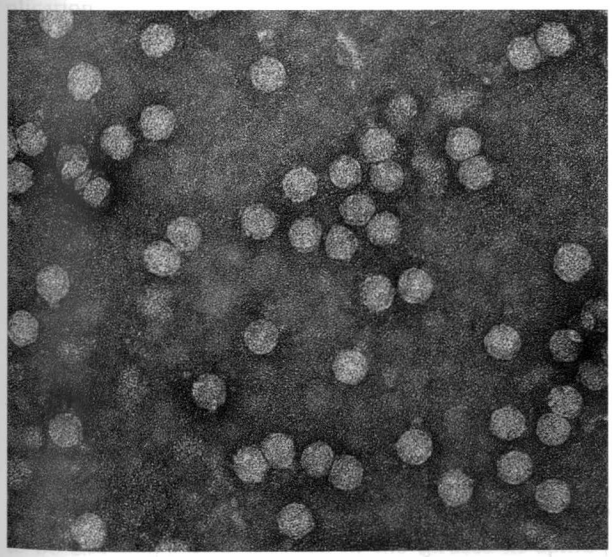

Figure 2 Transmission electron micrograph of soybean dwarf virus particles magnified 240 000×. Virions (stained with uranyl acetate) are *c.* 25 nm in diameter, hexagonal in appearance, and have no envelope.

genomes contain one or two small ORFs, ORFs 6 and 7, within or downstream of ORF5. An additional ORF, ORF8, has been discovered in ORF1 of PLRV. In the enamo- and poleroviruses ORF0 overlaps ORF1 by more than 600 nt, which also overlaps ORF2 by more than 600 nt. In the luteoviruses, ORF1 overlaps ORF2 by less than 50 nt. In all luteo- and polerovirus genome sequences (except for cucurbit aphid-borne yellows virus (CABYV) and GRAV), ORF4 is contained within ORF3. A single, in-frame amber (UAG) termination codon separates ORF5 from ORF3.

Luteovirids have relatively short 5′ and intergenic noncoding sequences. The first ORF is preceded by 21 nt in CABYV RNA and 142 nt in soybean dwarf virus (SbDV) RNA. ORFs 2 and 3 are separated by 112–200 nt of noncoding RNA. There is considerable variation in the length of sequence downstream of ORF5, which ranges from 125 nt for CYDV-RPV to 650 nt for SbDV.

Luteovirids employ an almost bewildering array of strategies to express their compact genomes. ORFs 0, 1, 2, and 8 are expressed directly from genomic RNA. Downstream ORFs are expressed from subgenomic RNAs (sgRNAs) that are transcribed from internal initiation sites by virus-encoded RNA-dependent RNA polymerases (RdRps) from negative-strand RNAs and are 3′-co-terminal with the genomic RNA. Since the initiation codon for ORF0 of polero- and enamoviruses is upstream of that of ORF1, translation of ORF1 is initiated by 'leaky scanning' in which ribosomes bypass

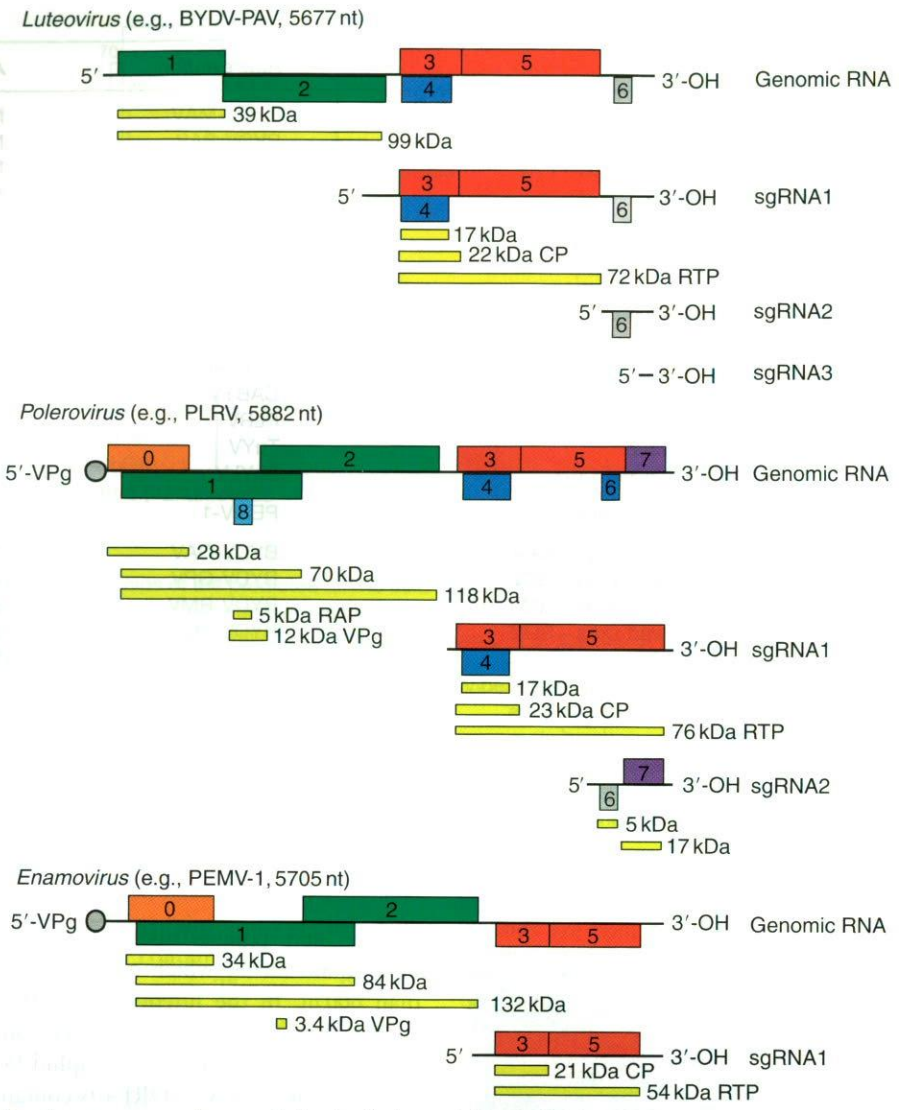

Figure 3 Maps of the virus genomes of genera in the family *Luteoviridae*. Individual ORFs are shown with open boxes. The ORFs are staggered vertically to show the different reading frames occupied by each ORF. The yellow boxes indicate protein products with the predicted sizes listed to the right of each. The polyproteins encoded by ORF1 of enamo- and poleroviruses contain the protease and the genome-linked protein (VPg). The predicted amino acid sequences of proteins encoded by ORF2 are similar to RNA-dependent RNA polymerases. ORF3, which encodes the major coat protein, is separated from ORF5 by an amber termination codon. ORF4, when present, is contained within ORF3 and encodes a protein required for virus cell-to-cell movement.

the AUG of ORF0 and continue to scan the genomic RNA until they reach the ORF1 AUG. The protein products of ORF2 are expressed as a translational fusion with the product of ORF1. At a low but significant frequency during the expression of ORF1, translation continues into ORF2 through a –1 frameshift that produces a large protein containing sequences encoded by both ORFs 1 and 2 in a single polypeptide. ORF8, which has only been identified in PLRV, resides entirely within ORF1 in a different reading frame and encodes a 5 kDa replication-associated protein. To express ORF8, sequences within the ORF fold into a structure called an internal ribosome entry site (IRES), which recruits ribosomes to initiate translation about 1600 nt downstream of the 5′ terminus of PLRV RNA.

ORFs 3, 4, and 5 are expressed from sgRNA1, the 5′ terminus of which is located about 200 nt upstream of ORF3 at the end of ORF2, and extends to the 3′ terminus of the genome. Luteo- and poleroviruses produce a second sgRNA that expresses ORFs 6 and 7. Luteoviruses produce a third sgRNA, which does not appear to encode a protein. ORF3 is translated from the 5′ terminus of sgRNA1. ORF4 of luteo- and poleroviruses, which encodes a 17 kDa protein, is contained within ORF3, and is expressed from the same sgRNA as ORF3 through a leaky scanning mechanism much like that used to express ORF1 in polero- and enamoviruses. In all luteovirids, ORF5 is expressed only as a translational fusion with the products of ORF3 by readthrough of the

UAG stop codon at the end of ORF3. This produces a protein with the product of ORF3 at its N-terminus and the product of ORF5 at its C-terminus.

While enamo- and polerovirus RNAs contain 5′ VPgs that interact with translation initiation factors, luteovirus RNAs contain only a 5′ phosphate. Unmodified 5′ termini are recognized poorly for translation initiation. To circumvent this problem, a short sequence located in the noncoding region just downstream of ORF5 in the BYDV-PAV genome acts as a potent enhancer of cap-independent translation by interacting with sequences near the 5′ termini of the genomic and sgRNAs to promote efficient translation initiation.

Research into the functions of the proteins encoded by luteovirids has shown that the 28–34 kDa proteins encoded by ORF0 are effective inhibitors of post-transcriptional gene silencing (PTGS). PTGS is an innate and highly adaptive antiviral defense found in all eukaryotes that is activated by double-stranded RNAs (dsRNAs), which are produced during virus replication. Consequently, viruses that contain mutations in ORF0 show greatly reduced accumulations in infected plants.

The ORF1-encoded proteins of enamo- and poleroviruses contain the VPg and a chymotrypsin-like serine protease that is responsible for the proteolytic processing of ORF1-encoded polyproteins. The protease cleaves the ORF1 proteins in *trans* to liberate the VPg, which is covalently attached to genomic RNA. The protein expressed by ORF8 of PLRV is required for virus replication. Luteovirid ORF2s have a coding capacity of 59–67 kDa for proteins that are very similar to known RdRps and hence likely represent the catalytic portion of the viral replicase.

ORF3 encodes the major CP of the luteovirids, which ranges in size from 21 to 23 kDa. ORF5 has a coding capacity of 29–56 kDa. However, ORF5 is expressed only as a translational fusion with the product of ORF3 when, about 10% of the time, translation does not stop at the end of ORF3 and continues through to the end of ORF5. The ORF5 portion of this readthrough protein has been implicated in aphid transmission and virus stability. Experiments with PLRV and BYDV-PAV have shown that the N-terminal region of the ORF5 readthrough protein determines the ability of virus particles to bind to proteins produced by endosymbiotic bacteria of aphid vectors. Interactions of virus particles with these proteins seem to be essential for persistence of the viruses in aphids. Nucleotide sequence changes within ORF5 of PEMV-1 abolish aphid transmissibility. The N-terminal portions of ORF5 proteins are highly conserved among luteovirids while the C-termini are much more variable.

The luteo- and polerovirus genomes possess an ORF4 that is contained within ORF3 and encodes proteins of 17–21 kDa. Viruses that contain mutations in ORF4 are able to replicate in isolated plant protoplasts, but are deficient or delayed in systemic movement in whole plants. Hence, the product of ORF4 seems to be required for movement of the virus within infected plants. This hypothesis is supported by the observation that enamoviruses lack ORF4. While luteo- and poleroviruses are limited to phloem and associated tissues, the enamovirus PEMV-1 is able to move systemically through other plant tissues in the presence of PEMV-2, which under natural conditions invariably coexists with PEMV-1.

Some luteo- and polerovirus genomes contain small ORFs within and/or downstream of ORF5. In luteoviruses, no protein products have been detected from these ORFs in infected cells. BYDV-PAV genomes that do not express ORF6 are still able to replicate in protoplasts. The predicted sizes of the proteins expressed by ORFs 6 and 7 of PLRV are 4 and 14 kDa, respectively. Based on mutational studies, it has been proposed that these genome regions may regulate transcription late in infection.

Evolutionary Relationships among Members of the *Luteoviridae*

Viruses in the family *Luteoviridae* have replication-related proteins that are similar to those in other plant virus families and genera. The luteovirus replication proteins encoded by ORFs 1 and 2 resemble those of members of the family *Tombusviridae*. In contrast, polymerases of poleroviruses and enamoviruses resemble those of viruses in the genus *Sobemovirus*. The structural proteins of some sobemoviruses also are similar to the major CP of luteovirids. Using an X-ray crystallography-derived structure of virions of the sobemovirus rice yellow mottle virus, which shares a CP amino acid sequence similarity of 33% with PLRV, it was possible to predict the virion structure of PLRV and other luteovirids.

Host Range and Transmission

Several luteovirids have natural host ranges largely restricted to one plant family. For example, BYDV and CYDV infect many grasses, BLRV infects mainly legumes, and CtRLV infects mainly plants in the family Apiaceae. Other luteovirids infect plants in several or many different families. For example, beet western yellows virus (BWYV) infects more than 150 species of plants in more than 20 families. As techniques for infecting plants with recombinant viruses have improved, the experimental host ranges of viruses have been expanded to include plants on which aphid vectors would not normally feed. For example, BYDV, CYDV, PLRV, and SbDV have been shown to infect *Nicotiana* species that had not been described previously as experimental hosts for the viruses when inoculated biolistically with viral RNA or using

Agrobacterium tumefaciens harboring binary plasmids containing infectious copies of the viruses. These results suggest that feeding preferences of vector aphids play important roles in defining luteovirid host ranges.

Luteovirids are transmitted in a circulative manner with varying efficiencies by at least 25 aphid species. With the exception of the enamovirus PEMV-1, members of the family *Luteoviridae* are transmitted from infected plants to healthy plants in nature only by the feeding activities of specific species of aphids. There is no evidence for replication of the viruses within aphid vectors. *Myzus persicae* is the most common aphid vector of luteovirids that infect dicots. Several different species of aphids transmit luteovirids that infect monocotyledenous plants (BYDV and CYDV) in a species-specific manner.

Circulative transmission of the viruses is initiated when aphids acquire viruses from sieve tubes of infected plants during feeding. The viruses travel up the stylet, through the food canal, and into the foregut (**Figure 4**). The viruses then are actively transported across the cells of the alimentary tract into the hemocoel in a process that involves receptor-mediated endocytosis of the viruses and the formation of tubular vesicles that transport the viruses through the epithelial cells and into the hemocoel. Luteovirids are acquired at different sites within the gut of vector aphids. PLRV and BWYV are acquired in the posterior midgut. BYDV, CYDV, and SbDV are acquired in the hindgut. CABYV is taken up at both sites. Viruses then passively migrate through the hemolymph to the accessory salivary gland where the viruses must pass through the membranes of the accessory salivary gland cells in a similar type of receptor-mediated transport process to reach the lumen of the gland. Once in the salivary gland lumen, viruses are expelled with saliva into vascular tissues of host plants. Since large amounts of virus can accumulate in the hemocoel of aphids, they may retain the ability to transmit virus for several weeks. Typically hindgut membranes are much less selective than those of the accessory salivary glands. Consequently, viruses that are not transmitted by a particular species of aphid often are transported across gut membranes and accumulate in the hemocoel, but do not traverse the membranes of the accessory salivary gland.

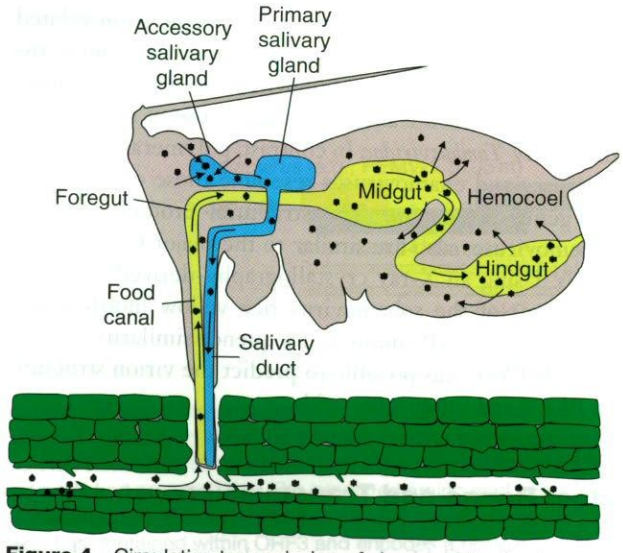

Figure 4 Circulative transmission of viruses of the family *Luteoviridae* by vector aphids. While feeding from sieve tubes of an infected plant, an aphid (shown in cross section) acquires virus particles, which travel up the stylet, through the food canal, and into the foregut. The virions are actively transported across cells of the posterior midgut and/or hindgut into the hemocoel in a process that involves receptor-mediated endocytosis. Virions then passively migrate through the hemolymph to the accessory salivary gland where they are again transported by a receptor-mediated process to reach the lumen of the gland. Once in the salivary gland lumen, the virions are expelled with the saliva into the vascular tissue of host plants. Aphids can retain the ability to transmit virus for several weeks. Hindgut membranes usually are much less selective than those of the accessory salivary glands, which is why viruses that are not transmitted by a particular species of aphid often accumulate in the hemocoel, but do not traverse the membranes of the accessory salivary gland.

The RTD of the minor capsid protein plays a major role in aphid transmission of luteovirids. The RTD interacts with symbionin produced by endosymbiotic aphid-borne bacteria, which may protect virions from degradation by the aphid immune system. The specificity of aphid transmission and gut tropism has been linked to the RTD in multiple luteovirids.

Unlike other luteovirids, PEMV-1 can be transmitted by rubbing sap taken from an infected plant on a healthy plant, in addition to being transmitted by aphids. This difference in transmissibility is dependent on its multiplication in cells co-infected with PEMV-2, but aphid transmissibility can be lost after several mechanical passages.

Replication

Luteovirids infect and replicate in sieve elements and companion cells of the phloem and occasionally are found in phloem parenchyma cells. PEMV-1 is able to move systemically into other tissues in the presence of PEMV-2. Virus infections commonly result in cytopathological changes in cells that include formation of vesicles containing filaments and inclusions that contain viral RNA and virions. The subcellular location of viral RNA replication has not been determined unequivocally. However, early in infection, negative-strand RNAs of BYDV-PAV are first detected in the nucleus and later in the cytoplasm, which suggests that at least a portion of luteovirus replication occurs in the nucleus. Synthesis of negative-strand RNA, which requires tetraloop structures at the 3′ end of BYDV-PAV genomic RNAs, is detected

in infected cells before the formation of virus particles. Late in infection, BYDV-PAV sgRNA2 inhibits translation from genomic RNA, which may promote a switch from translation to replication and packaging of genomic RNAs.

Virus–Host Relationships

While some infected plants display no obvious symptoms, most luteovirids induce characteristic symptoms that include stunting, leaves that become thickened, curled or brittle, and yellow, orange, or red leaf discoloration, particularly of older leaves of infected plants. These symptoms result from phloem necrosis that spreads from inoculated sieve elements and causes symptoms by inhibiting translocation, slowing plant growth, and inducing the loss of chlorophyll. Symptoms may persist, may vary seasonally, or may disappear soon after infection. Temperature and light intensity often affect symptom severity and development. In addition, symptoms can vary greatly with different virus isolates or strains and with different host cultivars.

Yield losses caused by luteovirids are difficult to estimate because the symptoms often are overlooked or attributed to other agents. US Department of Agriculture specialists estimated that yield losses from BWYV, BYDV, and PLRV infections were over $65 million during the period 1951–60. Plants infected at early stages of development by luteovirids suffer the most significant yield losses, which often are linearly correlated with the incidence of virus infection.

Epidemiology

Luteovirid infections have been reported from temperate, subtropical, and tropical regions of the world. Some of the viruses are found worldwide, such as BWYV, BYDV, and PLRV. Others have more restricted distribution, such as tobacco necrotic dwarf virus, which has been reported only from Japan, and groundnut rosette assistor virus, which has been reported only in African countries south of the Sahara.

Most luteovirids infect annual crops and must be reintroduced each year by their aphid vectors. Some viruses are disseminated in infected planting material, like PLRV where infected potato tubers are the principal source of inoculum for new epidemics. Consequently, programs to produce clean stock are operated around the world to control these viruses. Alate, that is, winged, aphid vectors may transmit viruses from local cultivated, volunteer, or weed hosts. Alternatively, alate aphids may be transported into crops from distant locations by wind currents. These vectors may bring the virus with them, or they may first have to acquire virus from locally infected hosts. The agronomic impact of luteovirid diseases depends both on meteorological events that favor movement and reproduction of vector aphids and susceptibility of the crop at the time of aphid arrival. Only aphid species that feed on a particular crop plant can transmit virus. Aphids that merely probe briefly to determine a plant's suitability will not transmit the viruses. Secondary spread of the viruses is often primarily by apterous, that is, wingless, aphids. The relative importance of primary introduction of virus by alate aphids and of secondary spread of virus by apterous aphids in disease severity varies with the virus, aphid species, crop, and environmental conditions.

Some members of the family *Luteoviridae* occur in complexes with other members of the family or with other plant viruses. For example, BYDV and CYDV often are found co-infecting cereals; BWYV and SbDV are often found together in legumes; and PLRV is often found co-infecting potatoes with potato virus Y and/or potato virus X. Some other plant viruses depend on luteovirids for their aphid transmission, such as the groundnut rosette virus, carrot mottle virus, and bean yellow vein banding virus (all umbraviruses), which depend on groundnut rosette assistor virus, carrot red leaf virus, and PEMV-1, respectively.

Diagnosis

An integral part of controlling luteovirid diseases is accurate diagnosis of infection. Because symptoms caused by luteovirids often resemble those caused by other biotic and abiotic factors, visual diagnosis is unreliable and other methods have been developed. Initially, infectivity, or biological, assays were used to diagnose infections. These techniques also have been used to identify species of vector aphids and vector preferences. In bioassays, aphids are allowed to feed on infected plants and then are transferred to indicator plants. These techniques are very sensitive, but can require several weeks for symptoms to develop on indicator plants. The strong immunogenicity of luteovirids has facilitated development of very specific and highly sensitive serological tests that can discriminate different luteovirids and sometimes even strains of a single virus species. Poly- and monoclonal antibodies for virus detection are produced by immunizing rabbits and/or mice with virus particles purified from infected plants. Techniques also have been developed to detect viral RNAs from infected plant tissues by reverse transcription-polymerase chain reaction (RT-PCR), which can be more sensitive and discriminatory than serological diagnostic techniques. Even so, serological tests are the most commonly used techniques for the detection of infections because of their simplicity and speed.

Control

Because methods are not available to cure luteovirid infections after diagnosis, emphasis has been placed on reducing losses through the use of tolerant or resistant plant cultivars and/or on reducing the spread of viruses by controlling aphid populations. Many luteovirids are transmitted by migrating populations of aphids that occur at similar times each year. For those virus–aphid combinations, it is sometimes possible to plant crops so that young, highly susceptible plants are not in the field when the seasonal aphid migrations occur. Insecticides have been used in a prophylactic manner to reduce crop losses. While insecticide treatments do not prevent initial infections, they can greatly limit secondary spread of aphids and therefore of viruses. In some instances biological control agents such as predatory insects and parasites have reduced aphid populations significantly. Genes for resistance or tolerance to infection by luteovirids have been identified in most agronomically important plant species infected by the viruses. For BYDV, PLRV, and SbDV, transgenic plants that express portions of the virus genomes have been produced through DNA-mediated transformation. In some cases, the expression of these virus genes in transgenic plants confers higher levels of virus resistance than resistance genes from plants.

See also: Barley Yellow Dwarf Viruses; Cereal Viruses: Wheat and Barley; Sobemovirus; Tombusviruses.

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Machlomovirus

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History

Maize chlorotic mottle virus (MCMV) was initially found in maize (*Zea mays*) and sorghum (*Sorghum bicolor*) fields in Peru in 1973. In 1976, the virus appeared in Kansas (USA) maize fields, alone and as part of a synergistic disease. In 1978, the second component of the synergistic disease, corn lethal necrosis (CLN), was identified as any maize-infecting virus of the family *Potyviridae*. MCMV and CLN spread to Nebraska, and both MCMV and CLN appeared in Mexico in 1982. On the island of Kauai, Hawaii (USA), a severe outbreak of MCMV appeared in the winter seed nurseries in 1989–90. MCMV is endemic in Peru and along the Kansas–Nebraska border. In 2004, MCMV and CLN were first detected in Thailand where MCMV continues to spread.

Taxonomy and Classification

The only known member of the genus *Machlomovirus* is the species *Maize chlorotic mottle virus*. Its inclusion in the family *Tombusviridae* is based on the high degree of homology of